REMARKS/ARGUMENTS

Claims 1-9 are pending in the application. Claims 1, 4, and 7 have been amended for clarification and to correct informalities identified in the Office Action. Support for the amendments can be found in the specification, particularly for example on page 13, line 2; page 2, line 24; and page 8, line 2. Claim 8 has been amended; support for this amendment can be found in the specification, particularly for example on page 13, lines 5-13. Applicants note that the specification has been amended to incorporate reference to sequence identifiers as requested, but additionally note that the indicated nucleic acids are not essential to the present application. No new matter has been added by way of amendment. Reexamination and reconsideration of the claims are respectfully requested.

The Invention

The inventors have identified the mechanisms that lead to maximum or minimum disease in a particular mouse strain in response to a given chlamydial inoculum. The identification of these mechanisms allows the rational selection of mouse strain, diet, and inoculum dose to provide a model for evaluating the progress of disease induced by chlamydial infection. Accordingly, the compositions and methods of the invention find use in studying the environmental and genetic factors affecting *Chlamydia*-induced disease as well as in evaluating the efficacy of therapeutic and prophylactic treatments of such disease, including diet and vaccination.

The Objections to the Claims Should Be Withdrawn

The Office Action (March 25, 2004, page 3, #5) has objected to claims 1d, 4, and 7 due to specified informalities. These claims have been amended to correct the informalities identified in the Action; accordingly, this objection has been obviated by amendment.

The Rejection of Claims Under 35 U.S.C. §112, First Paragraph, Should Be Withdrawn

The Office Action (March 25, 2004, page 3, #6) has rejected claims 1-9 under 35 U.S.C. §112, first paragraph because:

> [T]he specification, while being enabling for a method of evaluating the efficacy of Chlamydia-induced disease by varying the levels of NOS2 inhibitor or arginine in a mouse diet, does not reasonably provide enablement for a method of evaluating the efficacy of therapeutic or prophylactic treatment of Chlamydia-induced disease.

Applicants respectfully disagree with the conclusion that the specification does not provide enablement for a method of evaluating the efficacy of treatments of chlamydial disease.

As indicated in the quote above, the Office Action acknowledges that the specification is enabling for a method of evaluating chlamydial disease itself. Thus, the Action acknowledges that Applicants have provided a mouse lung disease model of infection and disease produced by *Chlamydia* spp. bacteria. Applicants agree with this conclusion. Further, Applicants emphasize that one of skill in the art, provided with a specification that is enabling for a method of evaluating a particular disease, would readily be able to use that method to evaluate the efficacy of a ***treatment*** for that disease.

The use of a provided animal model for the evaluation of treatments for a disease is well known in the art. Support for the idea that an animal model can be used for the evaluation of the efficacy of a therapeutic or prophylactic treatment is provided throughout the specification, particularly, for example, on pages 6 and 14. This state of the art is also demonstrated, for example, by Campbell *et al.* (1998) *Clin. Microbiol. Infect.* 4: 4523-32, cited in the Office Action. Campbell *et al.* report the use of a known apoE-deficient mouse strain to evaluate chlamydial disease and the role of macrophages in atherogenesis as well as to evaluate the use of roxithromycin for treatment of *C. pneumoniae* (page 4S30, col. II, top of column). Accordingly, Applicants respectfully submit that the rejection of claims for lack of enablement should be withdrawn.

The Rejections of Claims Under 35 U.S.C. §102 Should Be Withdrawn

The Office Action (March 25, 2004, page 7, #8) has rejected claims 1-9 under 35 U.S.C. §102(a) as anticipated by or, in the alternative, under 35 U.S.C. §103 as obvious over Huang *et al.* ((2002) *Proc. Nat'l. Acad. Sci. USA* 99: 3914-3919). Applicants are submitting herewith a declaration which should remove the Huang reference as prior art against the application.

As discussed in MPEP 2132.01 (citing *In re Katz*, 687 F.2d 450, 215 USPQ 14 (CCPA 1982)):

> Applicant's disclosure of his or her own work within the year before the application filing date cannot be used against him or her under 35 U.S.C. 102(a). . . . The rejection can be overcome by submission of a specific declaration by the applicant establishing that the article is describing applicant's own work.

As indicated on page 1 of the specification, the present application claims the benefit of U.S Provisional Application No. 60/401,070, filed August 5, 2002. The Huang reference has a publication date of March 19, 2002, which is less than one year before the effective filing date of the instant application. Furthermore, Applicants submit herewith a specific declaration by Applicant Bernhard Kaltenboeck, co-inventor on this application, which establishes that the Huang reference describes Applicants' own work. The additional six co-authors of this reference served as technical assistants, working under the direction and instructions of Applicant Kaltenboeck. Because it would be improper to reject claims 1-9 as anticipated by Applicants' own work under 35 U.S.C. §102(a), Applicants respectfully submit that this rejection of the claims should be withdrawn.

The Office Action (March 25, 2004, page 9, #9) has rejected claims 1-9 under 35 U.S.C. §102(b) as anticipated by Campbell *et al.* (1998) *Clin. Microbiol. Infect.* 4 (Supp. 4): S23-S32. Applicants respectfully traverse this rejection. Applicants note that claim 1 has been amended to clarify the scope of the invention. Particularly, clause (a) of claim 1 now recites the selection of a particular mouse strain "and identifying whether said strain is a low nitric oxide (NO) responder strain or a high NO responder strain...."

Campbell *et al.* discuss the use of an apoE-deficient mouse as an animal model to study the potential role for *Chlamydia pneumoniae* in atherogenesis. However, the Campbell reference neither teaches nor suggests the recited step of identifying whether a mouse strain is a low NO responder strain or a high NO responder strain. Accordingly, the Campbell reference cannot anticipate the present claim 1 or the other presently pending claims, all of which are dependent

on or incorporate the limitations of claim 1. For these reasons, Applicants respectfully request that the rejection of claims under 35 U.S.C. §102(b) over the Campbell reference be withdrawn.

The Office Action (March 25, 2004, page 10, #10) has rejected claims 1-9 under 35 U.S.C. §102(b) as anticipated by Yang *et al.* (1993) *Infect. Immun.* 4 (Supp. 4): S23-S32. Applicants respectfully traverse this rejection. Applicants note that claim 1 has been amended to clarify the scope of the invention. Particularly, clause (a) of claim 1 now recites the selection of a particular mouse strain "and identifying whether said strain is a low nitric oxide (NO) responder strain or a high NO responder strain...."

The Yang reference teaches the evaluation of various strains of mice as animal models for the study of *C. pneumoniae* infection and demonstrates that "six strains of commonly used laboratory mice were susceptible to intranasal inoculations with *C. pneumoniae.*" (p. 2040, col. I, first full paragraph) However, the Yang reference neither teaches nor suggests the recited step of identifying whether a mouse strain is a low NO responder strain or a high NO responder strain. Accordingly, the Yang reference cannot anticipate the present claim 1 or the other presently pending claims, all of which are dependent on or incorporate the limitations of claim 1. For these reasons, Applicants respectfully request that the rejection of claims under 35 U.S.C. §102(b) over the Yang reference be withdrawn.

Consideration Of Previously Submitted Information Disclosure Statement

Applicants note that the Office Action (3/25/04, page 3) stated that the IDS filed October 31, 2003 has been considered and that an initialed copy was enclosed with the Office Action. However, apparently there was some error during the mailing process, because no initialed copy was returned to Applicants with the Office Action. Accordingly, it is requested that an initialed copy of the Form 1449 that was submitted with Applicants' Information Disclosure Statement filed October 31, 2003 be forwarded to the undersigned with the next communication from the PTO. In order to facilitate review of the references by the Examiner, a copy of the Information Disclosure Statement and the Form 1449 are attached hereto. Copies of the cited references were provided at the time of filing the original Information Disclosure Statement, and, therefore, no

additional copies of the references are submitted herewith. Applicants will be pleased to provide additional copies of the references upon the Examiner's request if it proves difficult to locate the original references.

CONCLUSION

In view of the above amendments and remarks, Applicants submit that the rejections of the claims under 35 U.S.C. §§112, first and second paragraphs, and 102 are overcome. Applicants respectfully submit that this application is now in condition for allowance. Early notice to this effect is solicited.

If in the opinion of the Examiner, a telephone conference would expedite the prosecution of the subject Application, the Examiner is invited to call the undersigned.

It is not believed that extensions of time or fees for net addition of claims are required, beyond those that may otherwise be provided for in documents accompanying this paper. However, in the event that additional extensions of time are necessary to allow consideration of this paper, such extensions are hereby petitioned under 37 CFR § 1.136(a), and any fee required

therefore (including fees for net addition of claims) is hereby authorized to be charged to Deposit Account No. 16-0605.

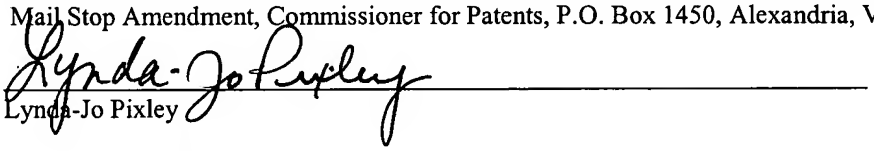
Respectfully submitted,

Leigh W. Thorne
Registration No. 47,992

Customer No. 00826
ALSTON & BIRD LLP
Bank of America Plaza
101 South Tryon Street, Suite 4000
Charlotte, NC 28280-4000
Tel Raleigh Office (919) 862-2200
Fax Raleigh Office (919) 862-2260

"Express Mail" mailing label number EL 387069299 US
Date of Deposit July 22, 2004

I hereby certify that this paper or fee is being deposited with the United States Postal Service "Express Mail Post Office to Addressee" service under 37 CFR 1.10 on the date indicated above and is addressed to:
Mail Stop Amendment, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450



Lynda-Jo Pixley

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Appl. No.: 10/632,426 Confirmation No.: 4998
Applicant(s): Kaltenboeck *et al.*
Filed: August 1, 2003
Art Unit: 1645
Examiner: Tammy K. Field
Title: MOUSE DISEASE MODEL FOR EVALUATION OF PROPHYLACTIC AND THERAPEUTIC TREATMENTS FOR CHLAMYDIA

Attorney Docket No.: 035721/265190
Customer No.: 00826

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

DECLARATION BY INVENTOR BERNHARD KALTENBOECK

I, Bernhard Kaltenboeck, hereby declare that:

1. I am a co-inventor, along with Jin Huang, of the invention claimed in the above-identified patent application, which claims priority to U.S. Provisional Patent Application No. 60/401,070, filed August 5, 2002.

2. I am a co-author of the manuscript entitled "The quantity of nitric oxide released by macrophages regulates *Chlamydia*-induced disease" which was published in *Proc. Nat'l. Acad. Sci. USA* 99: 3914-3919, dated March 19, 2002. Co-inventor Jin Huang is also a co-author on this manuscript. This manuscript discloses work that is subject matter of the above-identified application.

3. All but one of the other coauthors on the manuscript—Fred J. DeGraves, Stephen D. Lenz, Dongya Gao, Pu Feng, and Dan Li—were co-workers at the Department of Pathobiology in the College of Veterinary Medicine at Auburn University, Auburn, Alabama, at the time this manuscript was prepared. Tobias Schlapp was an employee of Bayer AG, Animal Health, Monheim, Germany, and is a coauthor on this manuscript. These individuals did not conceive of the subject matter claimed in the above-identified patent application and are not co-inventors thereof.

5. The role of the remaining six co-authors listed on the *Proc. Nat'l. Acad. Sci. USA* 99: 3914 manuscript was in the performance of technical services.

6. The *Proc. Nat'l. Acad. Sci. USA* 99: 3914 manuscript was published less than one year prior to August 5, 2002, which is the priority date of the above-identified patent application.

7. In view of the foregoing facts, I submit that the *Proc. Nat'l. Acad. Sci. USA* 99: 3914 manuscript is not prior art to the above-identified patent application.

8. All statements made herein of my own knowledge are true and all statements made on information and belief are believed to be true; and further, these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the above-identified patent application or any patent issued thereon.

July 19, 2004
Date

Bernhard Kaltenboeck

Commissioner For Patents
Alexandria, VA 22313

Date Mailed: October 28, 2003
Atty. Dkt. No. 35721/265190

Application No. 10/632,426; Filing Date August 1, 2003
Inventor(s): Kaltenboeck *et al.*; Title of Invention: "MOUSE DISEASE MODEL FOR EVALUATION OF PROPHYLACTIC AND THERAPEUTIC TREATMENTS FOR CHLAMYDIA"
Documents Enclosed: IDS (1 page); Form 1449 (3 page); and Copies of 42 References

Check Enclosed $0

Kindly acknowledge receipt by placing office stamp hereon and returning postcard to: **Lynda-Jo Pixley**
ALSTON & BIRD LLP

Attorney's Docket No. 35721/265190 PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re:	Kaltenboeck *et al.*	Confirmation No.:	Not yet assigned
Appl. No.:	10/632,426	Art Unit:	Not yet assigned
Filed:	August 1, 2003	Examiner:	Not yet assigned
For:	MOUSE DISEASE MODEL FOR EVALUATION OF PROPHYLACTIC AND THERAPEUTIC TREATMENTS FOR CHLAMYDIA		

October 28, 2003

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

INFORMATION DISCLOSURE STATEMENT
CITATION UNDER 37 C.F.R. § 1.97

Sir:

Attached is a list of documents on form PTO-1449. In accordance with the Office waiver published July 11, 2003, copies of the cited U.S. patents and patent application publications are not enclosed. Applicant does enclose copies of any cited foreign patent documents and non-patent literature in accordance with 37 CFR 1.98(a)(2).

It is requested that the Examiner consider these documents and officially make them of record in accordance with the provisions of 37 C.F.R. § 1.97 and Section 609 of the MPEP. By submitting the listed documents, Applicant in no way makes any admission as to the prior art status of the listed documents, but is instead submitting the listed documents for the sake of full disclosure.

Respectfully submitted,

W. Murray Spruill
Registration No. 32,943

CUSTOMER NO. 00826
ALSTON & BIRD LLP
Bank of America Plaza
101 South Tryon Street, Suite 4000
Charlotte, NC 28280-4000
Tel Raleigh Office (919) 862-2200
Fax Raleigh Office (919) 862-2260

CERTIFICATE OF MAILING

I hereby certify that this correspondence is being deposited with the United States Postal Service with sufficient postage as first class mail in an envelope addressed to:
Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450,
on October 28, 2003

Lynda-Jo Pixley

Substitute for form 1449/PTO
(Revised 04/2003)

INFORMATION DISCLOSURE STATEMENT BY APPLICANT

(Use as many sheets as necessary)

Sheet 1 of 3

Complete if Known	
Application Number	10/632,426
Filing Date	August 1, 2003
First Named Inventor	Kaltenboeck
Group Art Unit	Not yet assigned
Examiner Name	Not yet assigned
Attorney Docket Number	35721/265190

U. S. PATENT DOCUMENTS

Examiner Initials*	Cite No.	Document Number Number - Kind Code (if known)	Publication Date MM-DD-YYYY	Name of Patentee or Applicant of Cited Document	Pages, Columns, Lines, Where Relevant Passages of Relevant Figures Appear
	1	US-5,324,668	06-28-1994	Macri.	

FOREIGN PATENT DOCUMENTS

Examiner Initials	Cite No.	Foreign Patent Document Country Code - Number Kind Code (if known)	Publication Date MM-DD-YYYY	Name of Patentee or Applicant of Cited Document	Pages, Columns, Lines, Where Relevant Passages or Relevant Figures Appear	English Language Translation Attached

OTHER DOCUMENTS

Examiner Initials	Cite No.	Include name of the author (in CAPITAL LETTERS), title of the article (when appropriate), title of the item (book, magazine, journal, serial, symposium, catalog, etc.), date, page(s) , volume-issue number(s), publisher, city and/or country where published.	English Language Translation Attached
	2	ALLIONE, *et al.*, "Nitric Oxide Suppresses Human T Lymphocyte Proliferation Through IFN-γ-Dependent and IFN-γ-Independent Induction of Apoptosis," *The Journal of Immunology*, 1999, pp. 4182-4191, Vol. 163.	
	3	CHANG, *et al.*, "Arginase Modulates Nitric Oxide Production in Activated Macrophases," *Am. J. Physiol.*, 1998, pp. H342-H348.	
	4	DALTON, *et al.*, "Interferon γ Eliminates Responding CD4 T Cells during Mycobacterial Infection by Inducing Apoptosis of Activated CD4 T Cells," *J. Exp. Med.*, 2000, pp. 117-122, Vol. 192(1).	
	5	DETMERS, *et al.*, "Deficiency in Inducible Nitric Oxide Synthase Results in Reduced Atherosclerosis in Apolipoprotein E-Deficient Mice," *The Journal of Immunology*, 2000, pp. 3430-3435, Vol. 165.	
	6	DIEFENBACH, *et al.*, "Requirement for Type 2 NO Synthase for IL-12 Signaling in Innate Immunity," *Science*, 1999, pp. 951-955, Vol. 284.	
	7	GANTT, *et al.*, "Oxidative Responses of Human and Murine Macrophages During Phagocytosis of *Leishmania chagasi*," *The Journal of Immunology*, 2001, pp. 893-901, Vol. 167.	
	8	GOTOH, T. and MORI, M., "Arginase II Downregulates Nitric Oxide (NO) Production and Prevents NO-mediated Apoptosis in Murine Macrophage-derived RAW 264.7 Cells," *The Journal of Cell Biology*, 1999, pp. 427-434, Vol. 144, No. 3,	

Examiner Signature		Date Considered	

*Examiner: Initial if reference considered, whether or not citation is in conformance with MPEP 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to applicant.

Substitute for form 1449/PTO (Revised 04/2003)	Complete if Known	
INFORMATION DISCLOSURE STATEMENT BY APPLICANT *(Use as many sheets as necessary)*	Application Number	10/632,426
	Filing Date	August 1, 2003
	First Named Inventor	Kaltenboeck
	Group Art Unit	Not yet assigned
	Examiner Name	Not yet assigned
Sheet 2 of 3	Attorney Docket Number	35721/265190

	9	GUO, *et al.*, "Molecular Mechanisms of Increased Nitric Oxide (NO) in Asthma: Evidence for Transcriptional and Post-Translational Regulation of NO Synthesis," *The Journal of Immunology*," 2000, pp. 5970-5980, Vol. 164.	
	10	HERRICK, C.A. and BOTTOMLY, K., "To Respond or Not To Respond: T Cells in Allergic Asthma," *Nature Reviews/Immunology*, 2003, pp. 1-8, Vol. 3.	
	11	HOLLAND, *et al.*, "Conjunctival Scarring in Trachoma Is Associated with Depressed Cell-Mediated Immune Responses to Chlamydial Antigens," *The Journal of Infectious Diseases*, 1993, pp. 1528-1531, Vol. 168.	
	12	HU, *et al.*, "The Artherogenic Effects of Chlamydia are Eependent on Serum Cholesterol and Specific to *Chlamydia pneumoniae*," *Journal of Clinical Investigation.*, 1999, pp. 747-753, Vol. 103(5).	
	13	HUANG, *et al.*, "Nitric Oxide Regulates Th1 Cell Development Through the Inhibition of IL-12 Synthesis by Macrophages," *Eur. J. Immunol.*, 1998, pp. 4062-4070, Vol. 28.	
	14	HUANG, *et al.*, "IL-12 Administered During *Chlamydia psittaci* Lung Infection in Mice Confers Immediate and Long-Term Protection and Reduces Macrophage Inflammatory Protein-2 Level and Neutrophil Infiltration in Lung Tissue," *The Journal of Immunology*, 1999, pp. 2217-2226, Vol. 162.	
	15	HUANG, *et al.*, "The Quantity of Nitric Oxide Released by Macrophages Regulates *Chlamydia*-induced Disease," *PNAS*, 2002, pp. 3914-3919, Vol. 99(6).	
	16	IGIETSEME, *et al.*, "Resolution of Murine Chlamydial Genital Infection by the Adoptive Transfer of a Biovar-Specific, TH_1 Lymphocyte Clone," *Regional Immunology*, 1993, pp. 317-324, Vol. 5.	
	17	IGIETSEME, *et al.*, "Chlamydial Infection in Inducible Nitric Oxide Synthase Knockout Mice," *Infection and Immunity*, 1998, pp. 1282-1286, Vol. 66(4).	
	18	MORI, M. and GOTOH, T., "Relationship between Arginase Activity and Nitric Oxide Production," Chapter 12 , *Nitric Oxide Biology and Pathiobiology*, 2000, Chapter 12, pp.199-208.	
	19	JACKSON, *et al.*, "Specificity of Detection of *Chlamydia pneumoniae* in Cardiovascular Atheroma," *American Journal of Pathology*, 1997, pp. 1785-1790, Vol. 150(5).	
	20	KALTENBÖCK, *et al.*, "Genetically Determined Vigorous Innate Immunity is Associated with Protection Against Primary Chlamydial Lung Infection in Mice, but with Profound Disease Exacerbation in Reinfection," *Chlamydial Infections*, Proceedings of the Ninth International Symposium on Human Chlamydial Infection, June 21-26, 1998, pp. 403-406.	
	21	LYONS, *et al.*, "Molecular Cloning and Functional Expression of an Inducible Nitric Oxide Synthase from a Murine Macrophage Cell Line," *The Journal of Biological Chemistry*, 1992, pp. 6370-6374, Vol. 267(9).	
	22	MACMICKING, *et al.*, "Nitric Oxide and Macrophage Function," *Annu. Rev. Immunol.*, 1997, pp. 323-350, Vol. 15.	
	23	MAGEE, *et al.*, "*Chlamydia trachomatis* Pneumonia in the Severe Combined Immunodeficiency (SCID) Mouse," *Regional Immunology*, 1993, pp. 305-311, Vol. 5(6).	
	24	MILLS, *et al.*, "M-1/M-2 Macrophages and the Th1/Th2 Paradigm," *The Journal of Immunology*, 2000, pp. 6166-6173, Vol. 164.	
	25	MOAZED, *et al.*, "Evidence of Systemic Dissemination of *Chlamydia pneumoniae* via Macrophages in the Mouse," *The Journal of Infectious Diseases*, 1998, pp. 1322-1325, Vol. 177.	

Examiner Signature		Date Considered	

*Examiner: Initial if reference considered, whether or not citation is in conformance with MPEP 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to applicant.

Substitute for form 1449/PTO (Revised 04/2003)	Complete if Known	
	Application Number	10/632,426
	Filing Date	August 1, 2003
INFORMATION DISCLOSURE STATEMENT BY APPLICANT	First Named Inventor	Kaltenboeck
	Group Art Unit	Not yet assigned
(Use as many sheets as necessary)	Examiner Name	Not yet assigned
Sheet 3 of 3	Attorney Docket Number	35721/265190

	26	MOAZED, *et al.*, "*Chlamydia pneumoniae* Infection Accelerates the Progression of Atherosclerosis in Apolipoprotein E-Deficient Mice," *The Journal of Infectious Diseases*, 1999, pp. 238-241, Vol. 180.	
	27	MORRISON, *et al.*, "Gene Knockout Mice Establish a Primary Protective Role for Major Hisocompatibility Complex Class II-Restricted Responses in *Chlamydia trachomatis* Genital Tract Infection," *Infection and Immunity*, 1995, pp. 4661-4668, Vol. 63(12).	
	28	MUNDER, *et al.*, "Th1/Th2-Regulated Expression of Arginase Isoforms in Murine Macrophages and Dendritic Cells," *The Journal of Immunology*, 1999, pp. 3771-3777, Vol. 163.	
	29	OSWALD, *et al.*, "Low Response of BALB/c marophages to Priming and Activating Signals," *Journal of Leukocyte Biology*, 1992, pp. 315-322, Vol. 52.	
	30	PERRY, *et al.*, "Neither Interleukin-6 nor Inducible Nitric Oxide Synthase is Required for Clearance of *Chlamydia trachomatis* from the Murine Genital Tract Epithelium," *Infection and Immunity*, 1998, pp. 1265-1269, Vol. 66(3).	
	31	RAMSEY, *et al.*, "*Chlamydia trachomatis* Persistance in the Female Mouse Genital Tract: Inducible Nitric Oxide Synthase and Infection Outcome," *Infection and Immunity*, 2001, pp. 5131-5137, Vol. 69(8).	
	32	RANK, R.G., "Models of Immunity," *Chlamydia: Intracellular Biology, Pathogenesis, and Immunity*, 1999, Chapter 9, pp. 239-295.	
	33	ROSS, R., "Atherosclerosis – An Inflammatory Disease," *Mechanisms of Disease*, 1999, pp. 115-126, Vol. 340(2).	
	34	ROTTENBERG, *et al.*, "Role of Innate and Adaptive Immunity in the Outcome of Primary Infection with *Chlamydia pneumoniae*, as Analyzed in Genetically Modified Mice," *The Journal of Immunology*, 1999, pp. 2829-2836, Vol. 162.	
	35	SCHACHTER, J., "Infection and Disease Epidemiology," *Chlamydia: Intracellular Biology, Pathogenesis, and Immunity*, 1999, Chapter 6, pp. 139-169.	
	36	SCHWACHA, M.G. and EISENSTEIN, T.K., "Interleukin-12 is Critical for Induction of Nitric Oxide-Mediated Immunosuppression following Vaccination of Mice with Attenuated *Salmonella typhimurium*," *Infection and Immunity*, 1997, pp. 4897-4903, Vol. 65(12).	
	37	SCHWACHA, *et al.*, "*Salmonella typhimurium* Infection in Mice Induces Nitric Oxide-Mediated Immunosuppression through a Natural Killer Cell-Dependent Pathway," *Infection and Immunity*, 1998, pp. 5862-5866, Vol. 66(12).	
	38	STEVENSON, *et al.*, "Genetic Linkage of Resistance to *Listeria Monocytogenes* with Macrophage Inflammatory Responses," *The Journal of Immunology*, 1981, pp. 402-407, Vol. 127(2).	
	39	TEWS, J.K. and HARPER, A.E., "Tissue Amino Acids in Rats Fed Norlelucine, Norvaline, Homoarginine or Other Amino Acid Analogues," *J. Nutr.*, 1986, pp. 1464-1472, Vol. 116(8).	
	40	WILTSHIRE, *et al.*, "Genome-wide Single-nucleotide Polymorphism Analysis Defines Haplotype Patterns in Mouse," *PNAS*, 2003, pp. 3380-3385, Vol. 100(6).	
	41	WRIGHT, *et al.*, "Infectious Agents Are Not Necessary for Murine Atherogenesis," *J. Exp. Med.*, 2000, pp. 1437-1441, Vol. 191(8).	
	42	XIE, *et al.*, "Cloning and Characterization of Inducible Nitric Oxide Synthase from Mouse Macrophages," *Science*, 1992, pp. 226-228, Vol. 256.	

Examiner Signature		Date Considered	

*Examiner: Initial if reference considered, whether or not citation is in conformance with MPEP 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to applicant.